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Article:

Epidemiological implications of host biodiversity and vector biology: Key insights from simple models

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Abstract

Models used to investigate the relationship between biodiversity change and vector-borne disease risk often do not explicitly include the vector; they instead rely on a frequency-dependent transmission function to represent vector dynamics. However, differences between classes of vector (e.g. ticks and insects) can cause discrepancies in epidemiological responses to environmental change. Using a pair of disease models (mosquito- and tick-borne), we simulated substitutive and additive biodiversity change (where non-competent hosts replaced, or were added to, competent hosts, respectively), whilst considering different relationships between vector and host densities. We found important differences between classes of vector, including an increased likelihood of amplified disease risk under additive biodiversity change in mosquito models, driven by higher vector biting rates. We also draw attention to more general phenomena, such as a negative relationship between initial infection prevalence in vectors and likelihood of dilution, and the potential for a rise in density of infected vectors to occur simultaneously with a decline in proportion of infected hosts. This has important implications; the density of infected vectors is the most valid metric for primarily zoonotic infections, whilst the proportion of infected hosts is more relevant for infections where humans are a primary host.

Introduction

Vector-borne diseases (VBDs) account for significant proportions of global human mortality and morbidity (Hay et al. 2010; Bhatt et al. 2013; Campbell-Lendrum et al. 2015). Concerns have been growing that anthropogenic activities such as climate change, pollution, human migration, deforestation and encroachment into previously undisturbed habitats are exacerbating VBD risk in certain parts of the world (Harvell et al. 2002; Semenza and Menne 2009; Kilpatrick and Randolph 2012; Wesolowski et al. 2013; Parham et al. 2015). The consequences of changes in the community of animals acting as hosts for infectious disease, including VBDs, have received particular attention in recent years (*inter alia* Keesing et al. 2006, 2010; Pongsiri et al. 2009; Johnson et al. 2013; Wood et al. 2014).

The transmission of any vector-borne pathogen involves at least two other species - a vector and a host - and often many more (Auld and Tinsley 2015). For example, *Borrelia burgdorferi* sensu lato, which causes Lyme disease, is vectored by several different species of tick and is maintained by systemic infections in an array of hosts from three classes of vertebrate (Spielman et al. 1985; Matuschka et al. 1992). Altered abundance of any of these biotic components may cause changes in vector infection prevalence for a given disease, and therefore the risk of exposure for a given host, such as humans (Matuschka and Spielman 1986; Ostfeld and Keesing 2000). Links between biodiversity and individual disease risk are thus inevitable, though the consistency of the relationship is still debated (Randolph and Dobson 2012; Wood and Lafferty 2013; Ostfeld 2013; Ostfeld and Keesing 2013; Randolph and Dobson 2013; Lafferty and Wood 2013).

The ‘dilution effect’ has become the label for the process whereby an increase in biodiversity reduces the prevalence of a given infection. It has been studied in a large variety of disease systems, both vector-borne and directly transmitted, and in hosts from plant and animal kingdoms

(LoGiudice et al. 2003, 2008; Allan et al. 2009; Johnson et al. 2009; Haas et al. 2011; Young et al. 2013; Lacroix et al. 2014). When first described in the context of zoonotic VBDs, the key metric was the proportion of infected vectors (PIV) (Ostfeld and Keesing 2000), but the dilution effect literature has since broadened to include a range of metrics, in part because PIV is not as important as the density of infected vectors when considering human risk (Keesing et al. 2006; Ogden and Tsao 2009). It is, however, important to distinguish between the prevalence of a particular infection and the prevalence of disease in general. It is widely accepted, for example, that the diversity of pathogenic organisms (and therefore the overall disease burden in a community) is spatially correlated with biodiversity (Guernier et al. 2004; Wood et al. 2014). By contrast, the dilution effect applies specifically to the reduction in the prevalence of a single, given infection under conditions of host community diversification, and assumes that a diversifying community acquires at least one host that is poorer at transmitting the infection. Increased host biodiversity may be associated with dilution of one particular disease, but may also lead to increased prevalence of other diseases (Ostfeld and Keesing 2012; Randolph and Dobson 2012).

Nevertheless, some general rules regarding biodiversity-prevalence relationships for individual VBDs can be identified. Two fundamental factors are expected to influence epidemiological responses to host community change. The first factor is the transmission function, *i.e.* the mathematical relationship relating transmission to host density. The two basic functions are frequency-dependent (FD) and density-dependent (DD) (though there are important variants to consider; see Wonham et al. 2006). In FD transmission, the *per capita* force of infection is determined by the frequency of infected individuals; for VBDs, this effectively means that an individual host's chance of becoming infected is determined by the ratio of infected vectors to hosts. In DD transmission, the same individual's risk is simply a function of the absolute density of infected vectors. DD transmission is assumed to apply to directly-transmitted diseases such as influenza or measles, whereas FD transmission best describes sexually-transmitted infections such

as HIV, but is also typically applied to VBDs. Increased host diversity is held to dilute individual disease risk where transmission is FD (Rudolf and Antonovics 2005; Ostfeld and Keesing 2012; Mihaljevic et al. 2014), but when DD transmission is assumed, the outcome should depend on the nature of the host change (see below).

The second factor concerns the nature of the host community change. The simplest model of host community change is one where a community with a single species of competent host becomes a community with one competent and one non-competent host, or vice-versa. There are many ways in which this may occur, and we focus on the two scenarios that could be considered the extremes of a continuum: substitutive change, where host diversity increases without any change in overall host density; and additive change, where the addition of a second host species brings about a commensurate increase in overall density. Intuitively, dilution should occur after a substitutive increase in diversity (fig. 1A). When a proportion of reservoir-competent hosts is replaced by non-competent hosts, a similar proportion of vector bites is, in effect, wasted. This results in a lowered (diluted) prevalence of infection in the next generation of vectors (Matuschka and Spielman 1986). By contrast, if change is additive, the outcome is thought to depend on the transmission function; dilution with FD transmission, amplification with DD transmission (Rudolf and Antonovics 2005; Ostfeld and Keesing 2012; Mihaljevic et al. 2014).

One can also consider the numerical response of vectors to changes in host density. If host diversity is associated with host abundance, and if this subsequently determines vector population size, then any reduction of infection prevalence in vectors may be outweighed by increases in their absolute numbers (Dobson 2004; Ogden and Tsao 2009). There are four possible vector responses to an increase in host density: (i) no increase, (ii) a linear relationship between host and vector abundance, (iii) a less than linear relationship, (iv) a more than linear relationship. The likelihood of a particular response will depend on the biology of the vector. For example, all motile life stages

of ticks are parasitic, hence their populations are likely to be most strongly influenced by host abundance. For mosquitoes, the relationship between host and vector abundance appears to be more variable; survival of the non-biting larvae (as opposed to adult feeding success) often determines the dynamics of mosquito populations (Rueda et al. 1990; Reiter 2001; Pajjmans et al. 2007), but there are also many examples from the literature of host abundance driving adult mosquito abundance (e.g. McLaughlin and Vidrine 1988; Brown and Sethi 2002; Minikawa et al. 2002). Furthermore, the mobility of winged insects means that their probability of feeding within a given time period will be less limited by host density than that of ticks, because tick feeding success depends mainly on opportunistic contact with passing hosts (Randolph 2004). These differences in lifecycle and mobility mean that, typically, increases in host density are more likely to lead to an equivalent increase in vector density for ticks than for winged insects. For ticks in particular, this density increase could theoretically be more than linear - leading to an exponential increase in tick numbers - since each individual tick's chance of encountering a host increases with host density (Dobson and Randolph 2011). This should continue until high tick densities elicit host responses (e.g. grooming or immune reactions) that halt tick population growth (Randolph 1994; Kelly and Thompson 2000; Dobson et al. 2011). The nature of the numerical response influences the transmission function: an FD transmission function effectively approaches DD transmission if vectors respond more than linearly to an increase in host density, because the ratio of vectors to hosts (and therefore the bites per host) increases with host density (Ogden and Tsao 2009).

The numerical response is not, however, the only area in which vector biology can influence the epidemiological impact of changes in host community composition. Most biting insects take multiple, small blood-meals, whereas most ticks (especially ixodids) will typically take just three, large meals during their lifetimes (Randolph 1998). This has important implications for disease transmission: in the context of dilution, wasted bites matter far more for tick-borne than insect-borne pathogens. For these reasons, models of infectious disease transmission may not be fully

applicable to real-world VBDs if the vector is not explicitly modelled. Here, we demonstrate the role of vector biology in mediating the relationship between host biodiversity and disease prevalence using simple yet biologically realistic models. There is a rapidly growing empirical and theoretical literature on the dilution effect and related phenomena, and the interpretation of these studies will be aided by models that identify both the general and the idiosyncratic features of focal disease systems.

The Models

We present a pair of models of VBD transmission under conditions of vertebrate host community change: one for a generic mosquito-borne disease (MBD) and one for a generic tick-borne disease (TBD). These models are time-structured Leslie matrices that move in steps of three days for the mosquito model and 30 days for the tick model. Each model comprises a host and vector component that interact in order to calculate probabilities of mutual infection. For each model, we calculated two metrics: the change in density of infected vectors (DIV) and the change in proportion of competent hosts infected (PIH).

Mosquito model

Both competent and non-competent hosts have two life-stages: immature, lasting one year, and adult (fig. 2; Table 1). Adults reproduce once per year, and each has four offspring per reproductive event. Host life span is not fixed and instead follows an exponential distribution with a mean life span of approximately 12.8 months. Hosts move between ‘susceptible’, ‘infected’ and ‘recovered’ classes, and in initial simulations, recovered individuals cannot become re-infected (see section

‘Transmission success and host recovery’). The number of bites received by each host depends on the vector biting probability and the number of vectors per host. Infected hosts recover with probability R . The baseline adult vector-host ratio is set at 2.5:1; this corresponds to a daily per-host bite probability of ~ 0.83 (vectors are assumed to bite once per three-day timestep), in line with empirical observations of mosquito biting rates on humans (Mbogo et al. 2003). In the supplementary materials we also present data for model simulations where the initial ratios are 1.25:1 and 5:1.

The mosquito vectors have two discrete life-stages: a juvenile stage encompassing the transition from egg to larva to pupa, lasting twelve days, and an adult stage lasting thirty days (fig. 3). Adults bite a host and produce 50 eggs every three days, irrespective of host density, and bites are spread equally across available hosts. Bites on infective hosts infect the vector with probability determined by a variable probability of transmission success, and these vectors remain infected for life. We assume that male vectors do not bite, so matrix models explicitly follow only females (as a consequence, clutches effectively comprise 25 eggs). The following equations describe the calculation of numbers of newly-infected hosts following an infective vector bite:

$$b = iV/H \quad (1)$$

$$iH_{t+1} = sH_t * (1-(1-r)^b) \quad (2)$$

where b is the total number of infective bites received by each host per time-step, iV is the number of infective vectors, iH , sH and H are the numbers of infected, susceptible and all hosts, respectively, and r is the probability of transmission success. The following equation describes the equivalent process of calculating the number of infective vectors that result from bites by susceptible vectors on infected hosts:

$$iV_{t+1} = V_t * r * (iH/H) \quad (3)$$

where iV and V are the number of infected and all vectors, respectively. Note that, in these models, vector-to-host and host-to-vector transmission success probabilities are equal (Table 2).

Tick model

Host biology is identical to that in the mosquito model. The maximal value of nymphal ticks per host (during the seasonal peak of tick activity) is 50. This is the number of nymphs engorging per month. If it is assumed that the average nymph feeding event takes 3 days to complete, then this is equivalent to each host feeding a maximum of 5 nymphs at any given time, in line with empirical observations undertaken during peak tick activity periods (Duncan et al. 1978; Carpi et al. 2008; Matuschka et al. 1991). Data on larval and adult tick burdens are rare and unreliable (due to detectability and low numbers, respectively). So whilst we still model bites from larvae and adults, we calibrate the vector-host ratio by reference to nymphs only. In the supplementary materials we also present data for model simulations where the initial ratios are 2.5:1 and 10:1.

The tick vectors have three discrete life-stages: larva, nymph, adult, each lasting one year (Belozerov and Naumov 2002; Randolph et al. 2002 – see fig. 3). Larvae, nymphs and adults feed once per year, and bites are spread equally across available hosts. Adult females produce 2000 eggs after feeding, then die. We assume that larvae and nymphs of both sexes bite vertebrates, but that adult males do not (Randolph 1998). Bites on infective hosts infect the vector with probability determined by a variable probability of transmission success, and vectors remain infected for life. Vector feeding is seasonal, and peaks with juvenile host production (Randolph et al. 2002). This

seasonal feeding profile is achieved by moving a proportion of the free-living ticks of each stage (larvae, nymphs, adults) into respective separate ‘fed’ stages (see fig. 4), according to the following equation:

$$FX_{t+1} = X_t * p \quad (4)$$

where X is either larvae, nymphs or adults, and FX is the fed individuals of the respective stage. The coefficient p varies by month; in months 1-8, $p=0.5$, in month 9 $p=1$, and in months 10-12, $p=0$. This gradual release of unfed ticks into the fed stages is such that 99.6% are fed by month 8, and the peak is in month 1, which is the month when the hosts reproduce. The infection of hosts in the tick model follows that of the mosquito model (eqq. [1]–[2]). The infection of ticks by hosts is similar to equation (3), with the exception that the right-hand side is multiplied by p . Equation (4) therefore denotes the sum of fed ticks of each stage – both infected and uninfected.

In both models, the probability of mortality of all hosts and vectors are set to maintain population stability (Ogden and Tsao 2009). Results are insensitive to seeding conditions of infection prevalence in hosts and vectors; we start with an infection prevalence of 0.5 in both. Leslie matrices for all models are provided in the supplementary materials. Full R code for models, including graphics, is deposited in the Dryad Digital Repository: [http://dx. doi:10.5061/dryad.t24pq](http://dx.doi.org/10.5061/dryad.t24pq) (Dobson & Auld 2016).

Changes in host community composition

We investigated two types of biodiversity change in the host community: substitutive change, where half the competent hosts are replaced by non-competent individuals while host population size

remains constant; and additive change, where non-competent hosts are added such that the host population doubles. We considered each of these changes under three different numerical relationships (NR) between vectors and hosts: NR1, where vector abundance is independent of that of hosts, and remains constant; NR2, where vector abundance increases linearly with that of hosts; and NR3, where vector abundance increases more than linearly with that of hosts, such that an eventual doubling of the host population results in a quadrupling of the vector population. (The level of vector increase in NR3 was chosen to provide symmetry with the scenario of additive change with NR1, where host population size doubles as vector numbers remain constant).

Transmission success and host recovery

Transmission success probability varies between 0.1 and 1.0, and host recovery probability between 0 and 0.045 (mosquito models) and 0 and 0.45 (tick models). The differing scales are due to the different time-steps used in the two models; for comparison, the approximate mid-points 0.02 (mosquitoes) and 0.2 (ticks) result in 73.8% and 70.2%, respectively, of hosts recovering from infection within 6 months. Empirical recovery rates vary between combinations of pathogen and host (Gitau et al. 1999; Hofmeister et al. 1999; Gu et al. 2003). We chose a realistic spread of values, but acknowledge that certain hosts could clear infections at rates faster than we simulate. Within each model, the two community changes and three demographic relationships produce six possible scenarios. We present results for the 100 combinations of the 10 host recovery probabilities and 10 transmission success probabilities. As a default setting we assumed no vertical transmission (VT) in vectors or hosts, but we also present results showing effects of VT with a probability of 0.1 in hosts, vectors or both. In our basic host model, recovered hosts cannot be re-infected (SIR model). However, we also performed simulations with an SIRS host infection cycle. In the SIRS models, recovered hosts returned to the susceptible state one month after recovery. For

mosquito models, which move in three-daily time-steps, we adjusted the probability of moving from recovered to susceptible such that 99% of recovered hosts were susceptible within one month.

Simulations

All model runs were initiated with ten years of no host change to ensure infection equilibria had been reached, after which the host community change was introduced . Models were run for a further twenty years to ensure post-change equilibria were reached. Transmission success and host recovery probabilities were set at the start of each simulation. Infection parameters were taken as annual means from the final year of simulation (equilibrium always having been reached before this point).

Model assumptions

Some of the assumptions in these models reflect the general nature of the study, which is not designed to investigate the dynamics of any specific disease system. First, we do not assume any host preference among vectors. This may be unrealistic for most mosquitoes, meaning that our models may predict transmission interference – and hence dilution - too readily (though see Smith et al. 2007). For ticks, the assumption is more robust, since contact with hosts is often largely opportunistic; free-living ticks (as opposed to those that live in host burrows), in particular, feed on a wide variety of host species (Gray 1998). Even among vectors that are able to choose their hosts, host preference will not necessarily increase transmission to the most competent hosts, so the epidemiological consequences will not be universally consistent (though see Smallgame et al. 2013). Further simplifying assumptions are as follows: (i) Infection does not reduce host or vector

fitness. (ii) There is no ‘superinfection’, *i.e.*, the recovery of infected hosts is not delayed by subsequent infectious bites. (iii) Infection does not alter vector behaviour, although this does occur for some pathogen/vector combinations (Auld and Tinsley 2015, though see Cator et al. 2013). (iv) There are no density-dependent thresholds of vector-host ratio, though this can occur if hosts respond to high vector densities by increased grooming behaviour and/or immune response to bites (Levin and Fish 1998; Darbro and Harrington 2007; but see also Charlwood et al. 1995). Removal of this assumption would require knowledge of thresholds. Given the absence of supporting data, we assume no thresholds, and we do not simulate high numbers of vectors per host (see references above for empirical figures). If such responses were to occur, the result would be to cause a levelling-off in the relationship between vector density and strength of amplification at high vector densities. For our models, this would only affect the NR3 scenarios. (v) Per-vector biting probabilities are not limited at high or low host densities (the main characteristic of ‘reservoir frequency dependence’, a specific variant of FD transmission. See Wonham et al. (2006) for implications and further definitions). (vi) Host-to-vector and vector-to-host transmission success probabilities (the probability that contact between one infected and one non-infected individual results in infection of the latter) are equal.

Results

Change in density of infected vectors (DIV)

For both types of vector, epidemiological outcomes resulting from changes in host community composition depended strongly upon an interaction between the nature of host community change (additive or substitutive) and the numerical relationship between vector and host. When the host change was substitutive, DIV was always diluted. Under additive host change, dilution was most

likely where vector numbers were independent of host numbers (NR1), and where initial infection prevalence in vectors was lowest. Amplification was most likely where vector density increased at a greater rate than host density (NR3), and where infection prevalence was initially highest (figs 5 and 6). Amplification was also a more frequent outcome in mosquito models than tick models. This difference was maintained across all vector-host ratios tested (see supplementary materials figs S5-7 and S11-13).

Change in the proportion of infected hosts (PIH)

Dilution was more likely when measured as PIH as opposed to DIV, though at very high transmission success probabilities, dilution was either absent or negligible in all scenarios (fig. 7). Differences between vectors followed the pattern seen in DIV plots, and were maintained across all vector-host ratios tested (see supplementary materials figs S8-10 and S14-16).

Vertical transmission, re-infection and altered vector-host ratios

The inclusion of vertical transmission (VT) and host re-infection resulted in different disease outcomes in mosquito and tick models. Most differences between our sets of simulations are found in the plots showing the additive NR2 scenario, so for simplicity we focus on these plots (see supplementary material for figures for all VT and re-infection simulations, figs S21-28). Table 4 shows the number of cells in centre-bottom DIV and PIH plots that switched between dilution and amplification. Switches caused by VT were only recorded in DIV, and almost entirely found in the tick plots (44/45). All were of dilution switching to amplification (fig. 8). VT in hosts caused no switches. Host re-infection had a smaller impact, triggering only 5 switches among the four pairs of

plots (Table 4). Altering the vector-host ratio had predictable effects upon epidemiological parameters: rather than changing the character of the overall result, it instead shifted the threshold values of transmission success and recovery probabilities that determined the switch between amplification and dilution. The vector-host ratio was broadly proportional to the likelihood of amplification (see figures S5-S16 in supplementary material).

Discussion

Vector biology necessarily underpins VBD transmission (Randolph 1998), and yet it is given scant consideration in many disease models. Our simple yet general models incorporate important differences between ticks and mosquitoes in terms of their biting rates, reproductive biology, and responses to numeric changes in the host community. These differences confirm the importance of explicitly considering vectors in models of VBD, and provide more nuanced insights into the possible outcomes of environmental change.

VBD transmission is usually assumed to be frequency-dependent (e.g. Wonham et al. 2004; Joseph et al. 2013; Roche et al. 2013). However, some of the predictions that follow from the assumption of FD transmission may only hold for directly-transmitted diseases, and not VBDs (Dobson 2004). For example, whilst our NR3 scenario is underpinned by the same FD transmission term as the others, the function is essentially DD, since the ratio of vectors to hosts (and therefore the bites per host) increases with host density (Ogden and Tsao 2009).

The term ‘vector amplification’ is used to describe the situation whereby an increase in host abundance triggers a related rise in vector numbers (regardless of infection prevalence). Vector amplification can cause increases in overall vector density that balance – or in some cases outweigh

- any reductions in vector infection prevalence following biodiversity increase. Wood et al. (2014) use the term ‘spurious dilution’ to describe such reductions in infection prevalence that nonetheless do not reduce overall disease risk. The mechanics and implications of this phenomenon have been much-discussed elsewhere (e.g. Norman et al. 1999; Dobson 2004; Ostfeld and Keesing 2012; Randolph and Dobson 2012), and we do not dwell on it here. However, we emphasise the more subtle difference between vector amplification and the situation whereby vector abundance increases more than linearly with that of hosts (i.e. NR3; Randolph and Dobson 2012), shifting the transmission function from FD to DD.

Many authors have recognised that under additive host change, DD transmission should cause increased disease risk (e.g. Dobson 2004; Ostfeld and Keesing 2012). Our models highlight situations where amplification follows from more strictly FD transmission. In our mosquito model, additive host change and FD transmission causes amplification of DIV (under certain combinations of transmission success and host recovery probabilities). Previous models have suggested that amplification ought not to occur when transmission is FD, even when the host change is additive (Rudolf and Antonovics 2005; Mihaljevic et al. 2014). Indeed, if one considers only PIH (the prevalence of infection in hosts), it does not (note that the NR3 scenario is not strictly FD). However, an increase in DIV can occur because individual vectors bite both types of host. Bites on non-competent hosts are wasted, but the vector may still become infected later if it subsequently feeds on an infected host, in which case the earlier wasted bite is largely irrelevant. Consider, for simplicity, a vector that bites b hosts in its lifetime, and bites hosts at random in a population where there is a static host infection prevalence of x . The chance of the vector acquiring an infection during its lifetime is not x , but

$$x - (x - 1)^b$$

(5)

In reality, the value of x is not static, and instead will change as hosts recover or become infected, but it is nonetheless clear that the chance of any individual vector acquiring an infection must increase with the total number of hosts, b . A halving of the proportion of competent hosts does not, therefore, lead to a halving of infection prevalence in vectors (PIV; fig. 1B). In our models with FD transmission and additive change, dilution occurs only when transmission success falls below a certain threshold, because the probability that a wasted bite will prevent a vector from ever being infected declines as transmission success increases.

A consistent result to emerge from our models is that the higher the initial infection prevalence (correlated in these models with the product of transmission success probability and $1/(\text{host recovery probability})$), the less likely is dilution of DIV to result from either sort of biodiversity increase (additive or substitutive). When vectors bite several times, many bites will be from infected vectors on infected hosts. From an epidemiological perspective, these bites are equally ‘wasted’ as when infected vectors bite non-susceptible hosts (Smith et al. 2007). It follows, therefore, that the proportion of disease-carrying vectors may decline without affecting the rate at which susceptible hosts become infected. Susceptible hosts receive fewer bites, but as long as this number is above the threshold required to transmit an infectious dose of the pathogen, it is of little consequence. Similarly, if hosts rarely recover from infection, any genuinely wasted bites (infective bites on non-competent hosts) are less critical for maintenance of disease prevalence, because the total number of infective bites required to retain a reservoir of infectious hosts remains low. This finding is important because it implies that dilution occurs most readily where infection risk is low (i.e. low transmission success and rapid host recovery). Further, these results were robust to changes in the ability of recovered hosts to become re-infected.

Vertical transmission can provide an additional mechanism through which the effects of biodiversity change differ between MBDs and TBDs. The epidemiological consequences of vertical transmission were greater for tick-borne than for mosquito-borne infections. The mechanism underlying this is related to the differing rates of feeding: mosquitoes (in these models) will feed up to ten times, whereas the ticks can only feed three times (in line with real biological differences between the vectors; Randolph 1998). The mosquito therefore has more opportunities to acquire infection (see equation (1)), meaning that the potential for DIV and/or PIV to be increased by one extra infection opportunity (i.e. through maternal inheritance of the pathogen) is lower for mosquitoes than for ticks.

It follows that, all other factors being equal, the likelihood of dilution declines as the number of vectors per host decreases. From this observation one might assume dilution to be less likely in MBDs than in TBDs, since individual ticks typically bite fewer hosts than do individual mosquitoes (Randolph 1998). However, ticks may consistently differ in the way that they respond numerically to increases in host density. As discussed above, if a vector increases more than linearly with increases in host density, disease amplification becomes more likely. This scenario (NR3 in our models) is probably uncommon for mosquitoes, since their greater mobility (relative to ticks) means that their probability of encountering a host is less determined by host density, but is theoretically almost inevitable for ticks, at least below any thresholds of per-host tick loads that might cause a density-dependent response to parasitism in hosts (Ogden and Tsao 2009; Dobson 2014). Mosquitoes and related vectors are more likely to fit into the NR1 or NR2 scenario. However, whilst the NR1 response may be common among mosquitoes, dilution may not be as prevalent as our models suggest; the dilution predicted here is via transmission interference, which relies upon the assumption that vectors have no preference between competent and non-competent hosts. As already mentioned, mosquitoes may preferentially bite competent hosts, thereby reducing transmission interference and hence the potential strength of dilution.

Different metrics are used to describe disease outcomes in the literature, e.g. PIV, DIV, PIH (Salkeld et al. 2013). Our results demonstrate that these metrics are not interchangeable. The different implications of changes in PIV and DIV are frequently discussed, but we also reveal here the subtly divergent behaviour of DIV and PIH: it is possible for a rise in DIV to occur simultaneously with a decline in PIH, even without exponential vector population increase (compare ‘e’ panels in figs 5 and 7). Note that this is quite separate from what Wood et al. (2014) describe as ‘spurious dilution’ - the situation of declining PIV (not PIH) with increasing DIV, which is already well recognised (e.g., van Buskirk and Ostfeld 1995). The nature of the disease system will determine which metric is most relevant: where humans are one of the main hosts (e.g., malaria or dengue), a drop in PIH might be as important as a drop in DIV, but for zoonoses (where humans are only occasional, ‘spill-over’ hosts) such as Lyme borreliosis or West Nile Virus, PIH is only indirectly relevant. Our finding that that FD transmission need not prevent DIV amplification from occurring is particularly pertinent in this context.

Our modelling approach provides a generic framework for characterising the consequences of vector biology on the biodiversity-prevalence relationship. We did not aim to produce a set of specific predictions for combinations of disease systems and conditions under which dilution or amplification ought to occur – our aim was to instead build a foundation upon which more tailored models can be built to explore individual VBD systems. To this end, we have deliberately avoided providing quantitative results, as doing so would be claiming a false accuracy. We also remind readers that ‘additive’ and ‘substitutive’ host changes lie at extremes of a continuum, and that disease outcomes in complex communities are unlikely to follow simple rules (e.g., Roche et al. 2013; Mihaljevic et al. 2014). Our results have, however, clearly demonstrated that modelling FD transmission without explicitly considering the vector can limit model applicability to real-world VBD systems.

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Tables

Table 1. Parameters for the host components of models.

Parameter	Definition	Value	
		<i>Mosquito model</i>	<i>Tick model</i>
R	Probability of recovery	0 - 0.045	0 - 0.45
F	Probability of re-infection	0 , 0.631	0 , 1
r	Transmission success probability	0.1 - 1	0.1 - 1
N	Number of vectors	(Variable)	(Variable)
H	Number of hosts	(Variable)	(Variable)
m	Ratio of vectors to hosts	N/H	N/H
iN	Number of infected vectors	(Time-dependent)	(Time-dependent)
a	Vector infection prevalence	iN/N	iN/N
b	Bites per vector per timestep	1	(Dependent upon monthly feeding proportion, q)
c	Infected bites received per host per timestep	a.m.b	a.m.q
sti _H	Probability of change from susc. to inf. host	1-((1-r) ^c)	1-((1-r) ^c)
JS	Juvenile survival per timestep	0.99217	0.92224
AS	Adult survival per timestep	0.99217	0.92224
vt	Probability of vertical transmission	(Variable)	(Variable)
		<i>Both models</i>	
		<i>On annual timesteps</i>	<i>All other timesteps</i>
sJS	Susceptible juvenile survival	0	JS.(1-sti _H)
iJS	Infected juvenile survival	0	JS.(1-R)
rJS	Recovered juvenile survival	0	JS.(1-f)
sAS	Susceptible adult survival	0	AS.(1-sti _H)
iAS	Infected adult survival	0	AS.(1-R)
rAS	Recovered adult survival	AS	AS.(1-f)
sJtA	Susceptible juvenile to susceptible adult	JS.(1-sti _H)	0
iJtA	Infected juvenile to infected adult	JS.(1-R)	0
rJtA	Recovered juvenile to recovered adult	JS	0
sJiJ	Susceptible juvenile to infected juvenile	0	JS.sti _H
sJiA	Susceptible juvenile to infected adult	JS.sti _H	0
sAiA	Susceptible adults to infected adults	0	AS.sti _H
iArA	Infected adults to recovered adults	0	AS.R
iJrJ	Infected juvenile to recovered juvenile	0	JS.R
iJrA	Infected juvenile to recovered adult	JS.R	0
rJsJ	Recovered juvenile to susceptible juvenile	0	JS.f
rJsA	Recovered juvenile to susceptible adult	JS.f	0
rAsA	Recovered adult to susceptible adult	AS.f	AS.f
HP	Offspring per host per year	4	0
iHPs	Susceptible offspring per infected host per year	4.(1-vt)	0
iHPi	Infected offspring per infected host per year	4.vt	0
ncJS	Juvenile survival, non-competent host	0	JS
ncJtA	Juvenile to adult, non-competent host	JS	0
ncAS	Adult survival, non-competent host	AS	AS

Table 2. Vector parameters for the mosquito model.

Parameter	Definition	Value
r	Transmission success probability	0.1 - 1
iH	Number of infected hosts	(Time-dependent)
H	Number of hosts	(Variable)
b	Bites per vector per timestep	1
sti _v	Probability of change from susc. to inf. vector	b.r.(iH/H)
vt	Probability of vertical transmission	0 , 0.1
VS	Stage survival per timestep	0.39450503
StoSI	Susceptible adults to infected adults	sti _v .VS
StoSA	Stage survival (susceptible adults)	(1-sti _v).VS
VP	Offspring per vector per bloodmeal	50
iVPi	Infected offspring per infected vector per bloodmeal	VP.vt
iVPs	Susceptible offspring per infected vector per bloodmeal	VP.(1-vt)

Table 3. Vector parameters for the tick model.

Parameter	Definition	Value	
p	Monthly feeding proportion	Months 1-8: 0.5; month 9: 1; months 10-12: 0	
r	Transmission success probability	0.1 - 1	
H	Number of hosts	(Variable)	
iH	Number of infected hosts	(Variable)	
E	Eggs per adult female per year	2000	
ES	Egg survival per month	$((1/1000)^{(1/3)})^{(1/12)}$	
LS	Larva survival per month	$((1/1000)^{(1/3)})^{(1/12)}$	
NS	Nymph survival per month	$((1/1000)^{(1/3)})^{(1/12)}$	
AS	Adult survival per month	$((1/1000)^{(1/3)})^{(1/12)}$	
stiv	Probability of change from susc. to fed infected vector	$r \cdot (iH/H) \cdot p$	
stnv	Probability of change from susc. to fed uninfected vector	$(1 - r \cdot (iH/H)) \cdot p$	
vt	Probability of vertical transmission	0, 0.1	
		<i>On annual time-steps [time-steps]</i>	<i>All other time-steps</i>
iES	Infected egg survival	0 [12]	ES
sES	Susceptible egg survival	0 [12]	ES
iLS	Infected larva survival	LS.(1-p)	LS.(1-p)
sLS	Susceptible larva survival	LS.(1-p)	LS.(1-p)
iNS	Infected nymph survival	NS.(1-p)	NS.(1-p)
sNS	Susceptible nymph survival	NS.(1-p)	NS.(1-p)
iAS	Infected adult survival	AS.(1-p)	AS.(1-p)
sAS	Susceptible adult survival	AS.(1-p)	AS.(1-p)
iFLS	Fed, infected larva survival	0 [12]	LS
sFLS	Fed, uninfected larva survival	0 [12]	LS
iFNS	Fed, infected nymph survival	0 [12]	NS
sFNS	Fed, uninfected nymph survival	0 [12]	NS
iFAS	Fed, infected adult survival	0 [10,11,12]	AS
sFAS	Fed, uninfected adult survival	0 [10,11,12]	AS
iEiL	Infected egg to infected larva	ES [12]	0
sEsL	Susceptible egg to susceptible larva	ES [12]	0
sLiF	Susceptible larva to fed, infected larva	0 [12]	LS.stiv
sLsF	Susceptible larva to fed, uninfected larva	0 [12]	LS.stnv
iLiF	Infected larva to fed, infected larva	0 [12]	LS.p
iFiN	Fed, infected larva to infected nymph	LS [12]	0
sFsN	Fed, uninfected larva to susceptible nymph	LS [12]	0
sNiF	Susceptible nymph to fed, infected nymph	0 [12]	NS.stiv
sNsF	Susceptible nymph to fed, uninfected nymph	0 [12]	NS.stnv
iNiF	Infected nymph to fed, infected nymph	0 [12]	NS.p
iFiA	Fed, infected nymph to infected adult	NS [12]	0
sFsA	Fed, uninfected nymph to susceptible adult	NS [12]	0
sAiF	Susceptible adult to fed, infected adult	0 [10,11,12]	AS.stiv
sAsF	Susceptible adult to fed, uninfected adult	0 [10,11,12]	AS.stnv
iAiF	Infected adult to fed, infected adult	0 [10,11,12]	AS.p
iVPi	Infected eggs per infected vector	AS.E.vt [10]	0
iVPs	Susceptible eggs per infected vector	AS.E.(1-vt) [10]	0
sVP	Susceptible eggs per susceptible vector	AS.E [10]	0

Table 4. Impacts of the addition of vertical transmission in vectors (top two rows) and re-infection of recovered hosts (bottom two rows) for plots of DIV and PIH in mosquito and tick models.

Cells switching in DIV plot			Cells switching in PIH plot	
	Dilution to Amplification	Amplification to Dilution	Dilution to Amplification	Amplification to Dilution
<i>Impact of vertical (vectorial) transmission</i>				
Mosquito	1	0	0	0
Tick	44	0	0	0
<i>Impact of host re-infection</i>				
Mosquito	2	0	0	0
Tick	2	1	0	0

Figure legends.

Figure 1: *A*, Dilution in a vector-borne disease. Substituting non-competent hosts for competent ones reduces the prevalence of infection in vectors accordingly. *B*, A schematic that incorporates multiple vector feeding. The ‘wasted’ bites on non-competent hosts will, to an extent, be nullified by subsequent bites on competent, infected hosts. The actual vector infection prevalence will fall between 50 and 100%.

Figure 2: Flow diagram for competent host model, which is a stage-classified matrix model. In mosquito models, individuals move between stages every three days; in tick models, individuals move monthly. The stages (in circles) sJ, sA, iJ, iA, rJ and rA correspond to susceptible juvenile, susceptible adult, infected juvenile, infected adult, recovered juvenile, recovered adult, respectively. The non-competent host model comprises only the two ‘susceptible’ age classes. Definitions and values of the coefficients may be found in Table 1.

Figure 3: Flow diagram for the mosquito model. Individuals move between stages every three days. The stages (in circles) beginning iL, iA, sL, and sA correspond to infected larvae, infected adults,

susceptible larvae and susceptible adults, respectively. The numbers denote the age class (each age class lasts three days); mosquitoes in this model therefore survive for (4×3) 12 days as larvae and (10×3) 30 days as adults. Definitions and values of the coefficients may be found in Table 2.

Figure 4: Flow diagram for the tick model. Individuals move between stages every month. The stages (in circles) iE, iL, iN, iA, sE, sL, sN and sA correspond to infected eggs, larvae, nymphs and adults, and susceptible eggs, larvae, nymphs and adults, respectively. Definitions and values of the coefficients may be found in Table 3.

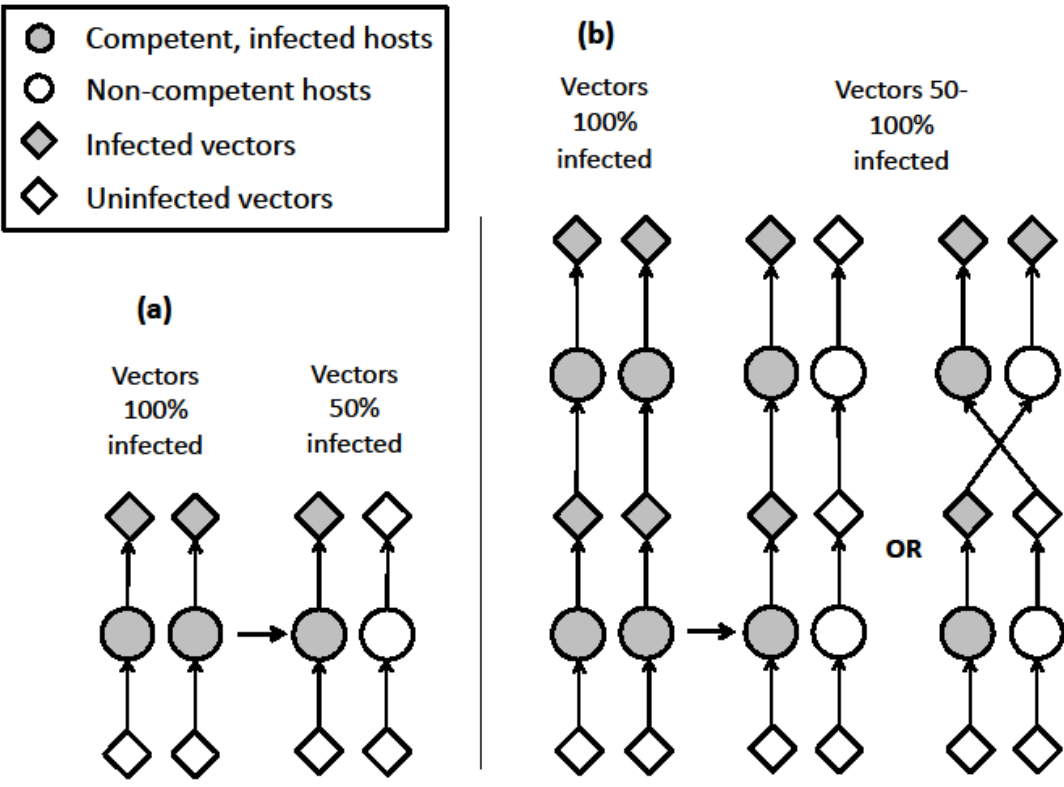
Figure 5: Change in density of infected vectors (DIV) in the mosquito model after increase in host community diversity, either substitutively or additively. Numerical responses in vectors to changes in overall host population size are denoted by the top row of plots. Blue cells show where DIV decreased after the host change; red cells show where it increased. The point size for the top-left plot represents the initial proportion of infected adult vectors.

Figure 6: Change in density of infected vectors (DIV) in the tick model after increase in host community diversity, either substitutively or additively. (All details are as per fig.5).

Figure 7: Change in proportion of infected competent hosts (PIH) in the mosquito model after increase in host community diversity, either substitutively or additively. Numerical responses in vectors to changes in overall host population size are denoted by the top row of plots. Purple cells show where PIH decreased after the host change; orange cells show where it increased. The point size for the top-left plot represents the initial proportion of infected hosts.

Figure 8: Impact of vertical (within vectors) transmission on change in density of infected vectors: Upper plots: Mosquitoes; comparison of panel 'e' from fig. 5 (left) with the same output from a model with vertical transmission in vectors with a probability of 0.1 (right). Lower plots: as previous, for ticks (fig. 6, panel 'e').

FIGURES



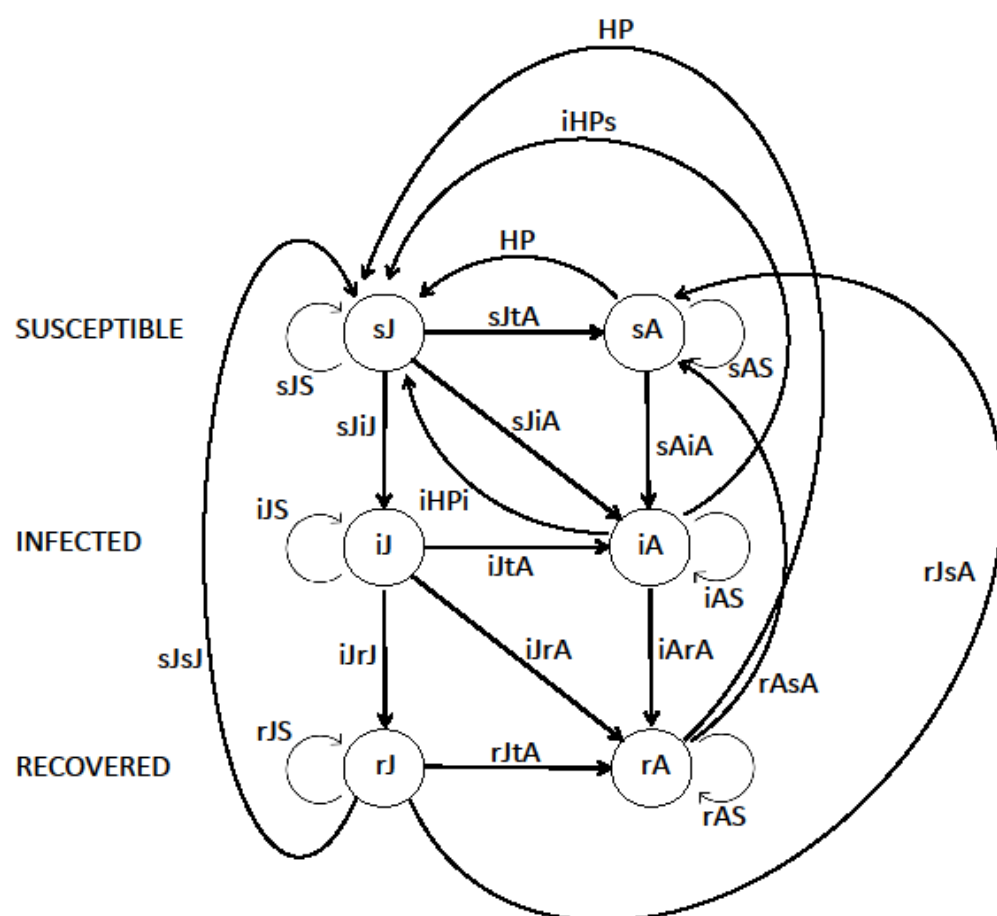


Figure 2.

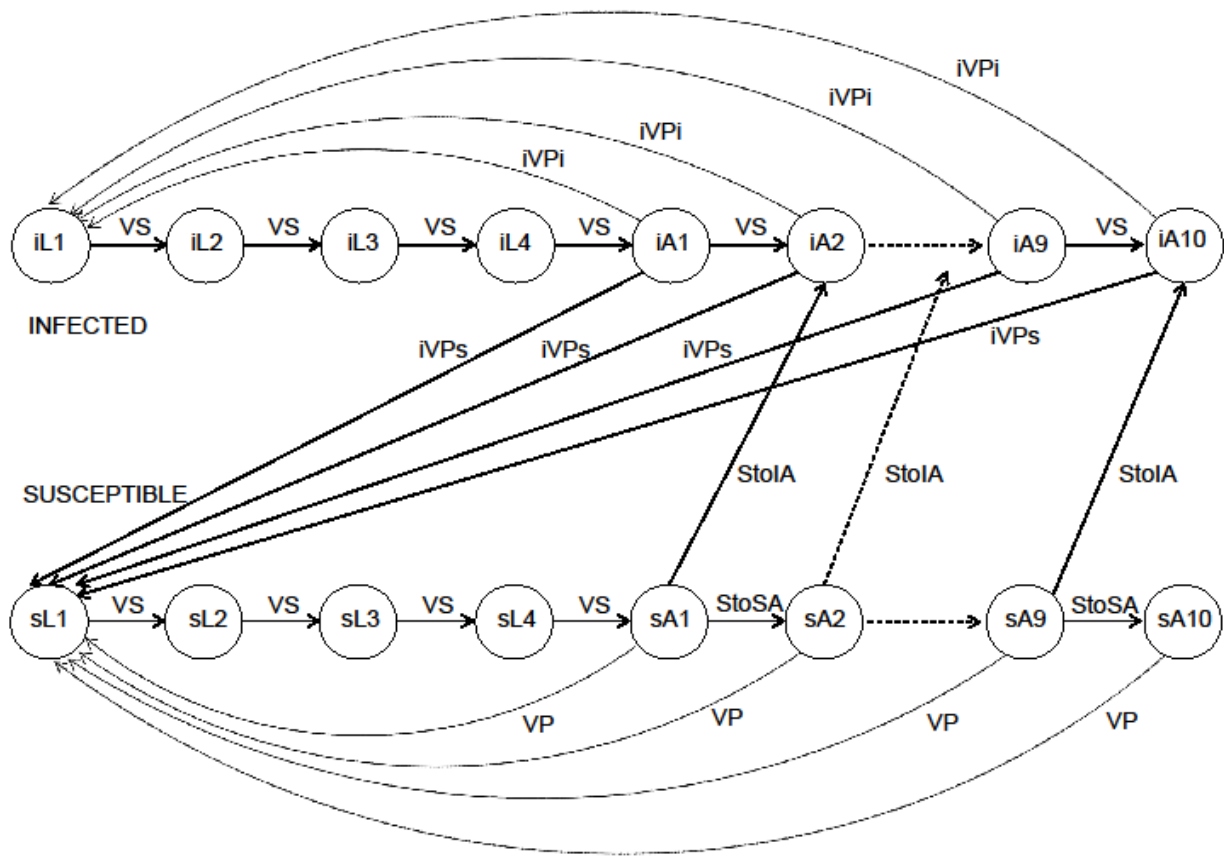


Figure 3.

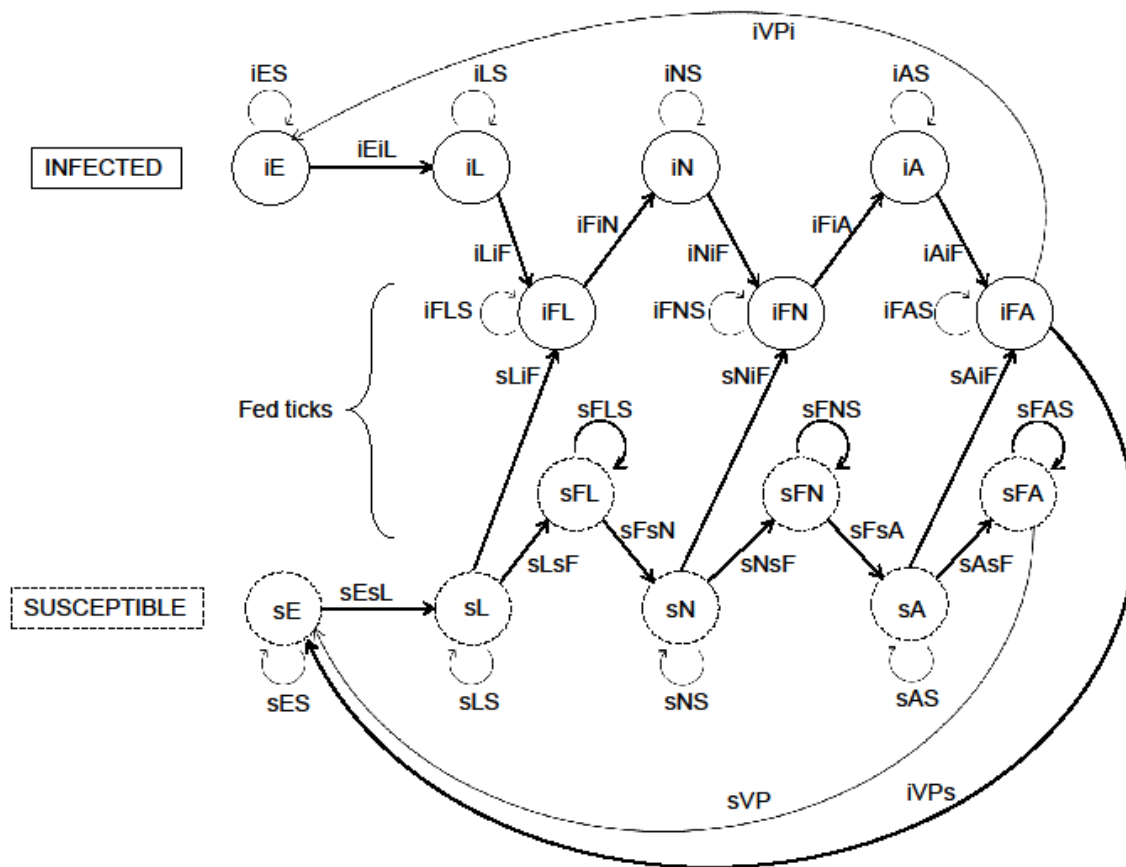


Figure 4.

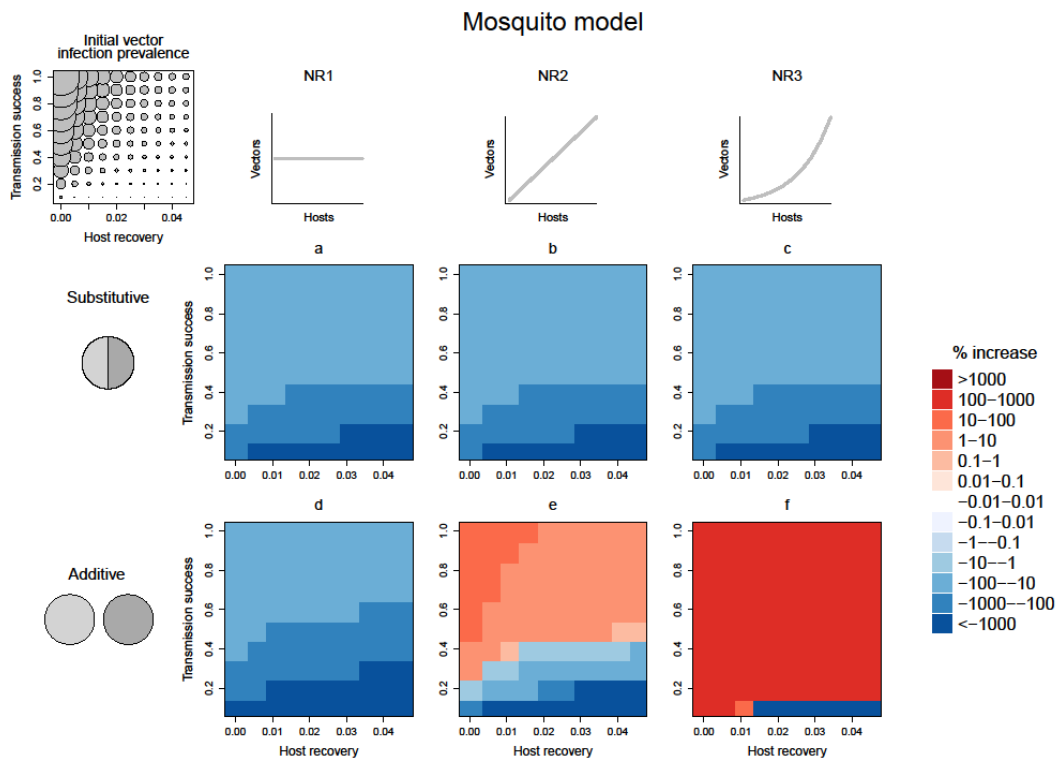


Figure 5.

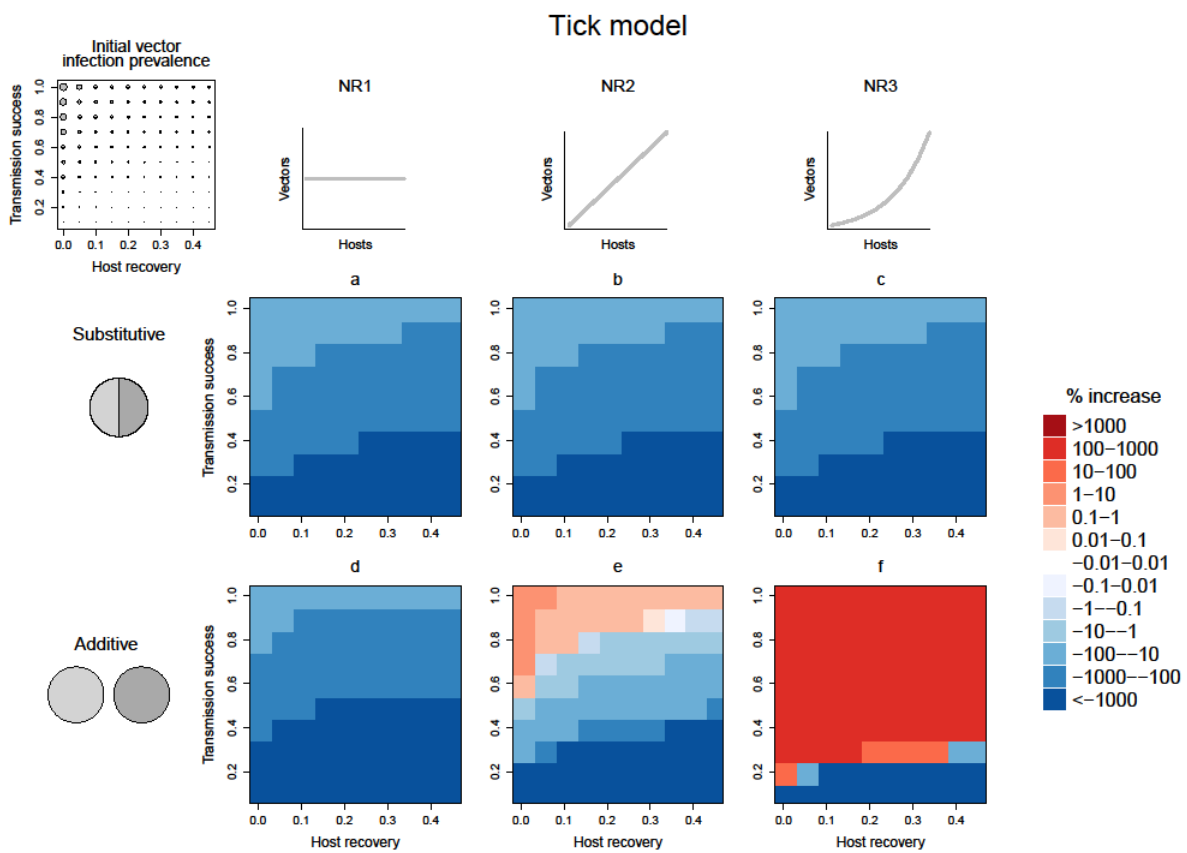


Figure 6.

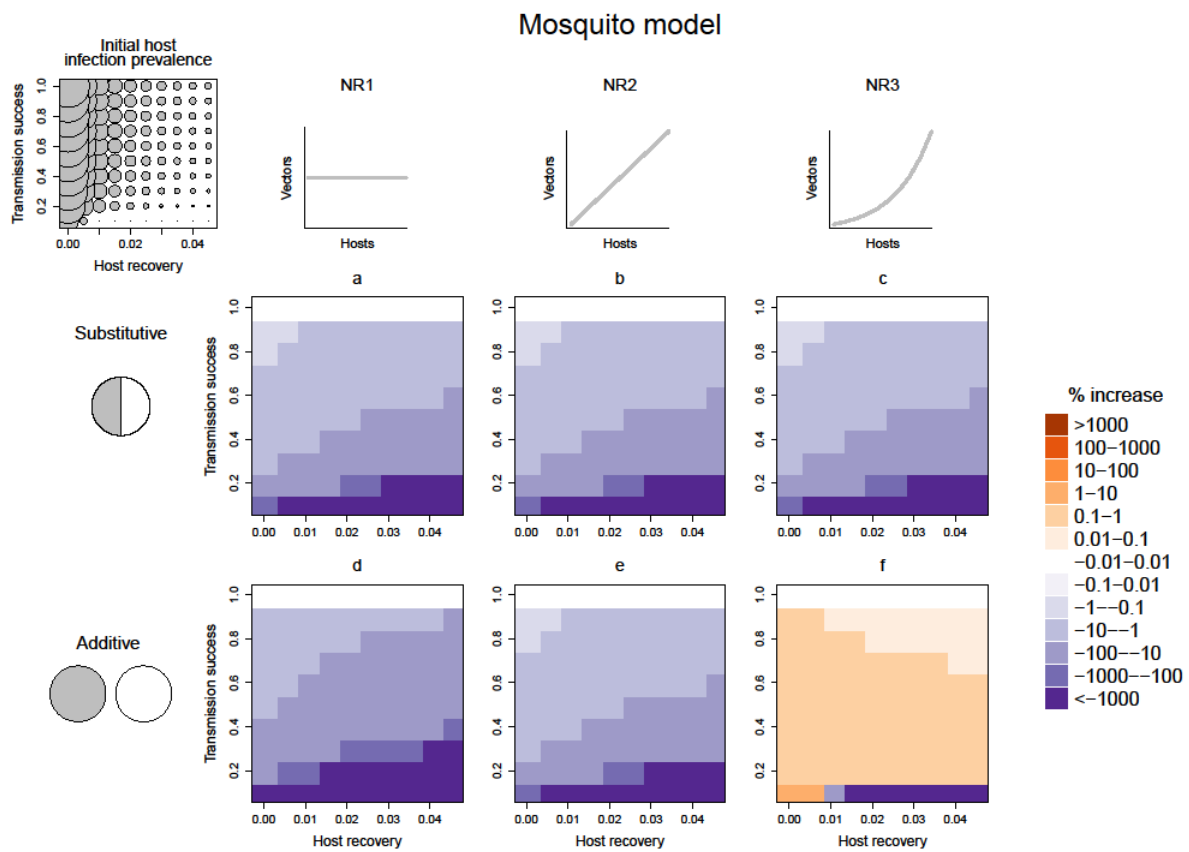


Figure 7.

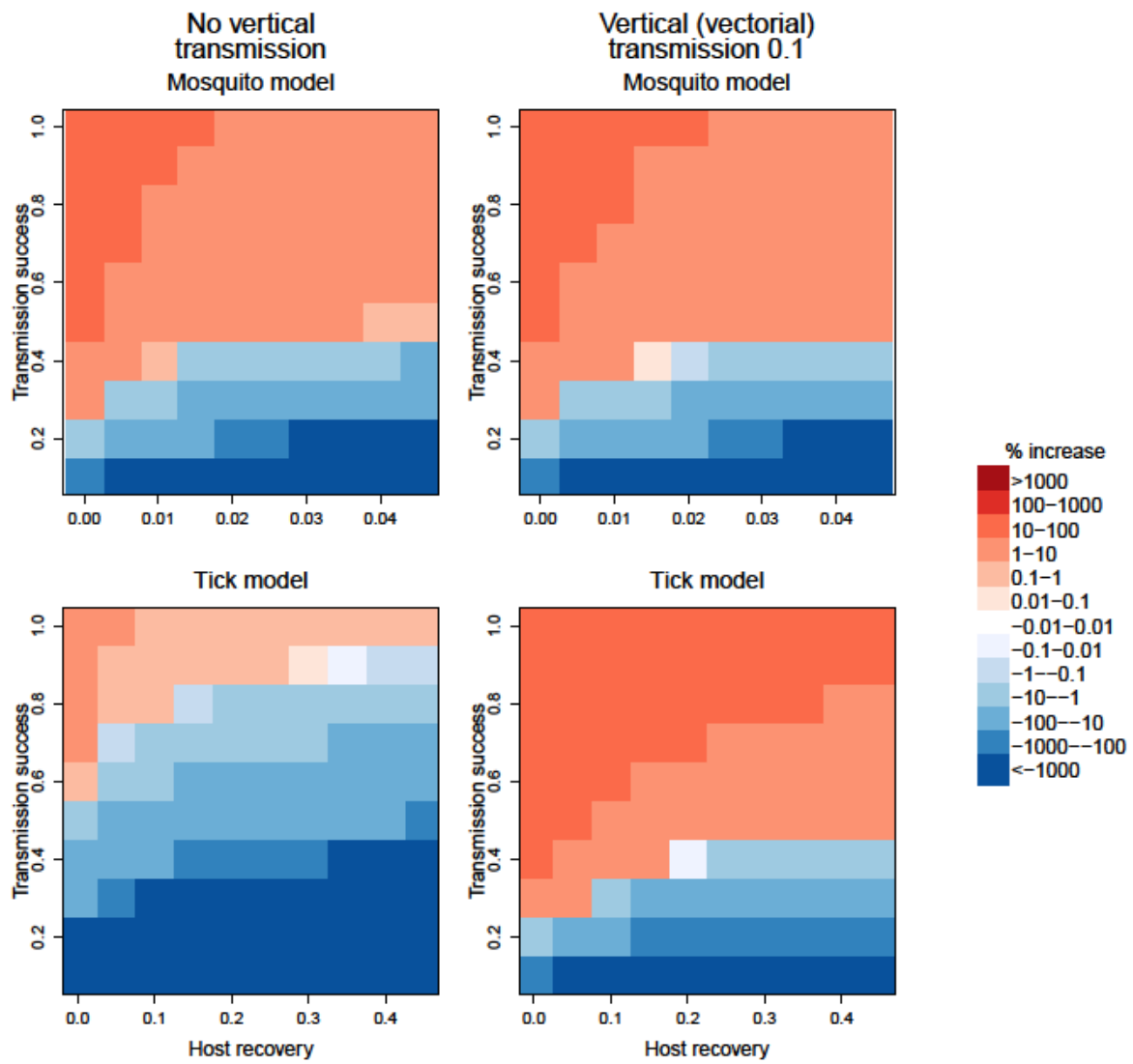


Figure 8.